mivacurium may be acceptable. Neither drug can be considered a full replacement for succinylcholine, however.

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Preparing Children for Anesthesia and Surgery

IN PREPARING A CHILD for surgery, an anesthesiologist is faced with the unenviable task of separating a starved and frightened child from anxious parents. The scientific rationale for a lengthy preoperative fast has recently been questioned, and based on new studies, preoperative fasting guidelines have been modified. In addition, newer hypnotic drugs and modes of administration can ease the separation of children from their parents.

Whereas the tradition of a preoperative fast can be traced to 1858, decades of studying the epidemiology and risk factors for perianesthetic aspiration of stomach contents have not determined the proper duration of the fast. It has now been established that the gastric fluid of children (older than 1 year) who are allowed to drink clear liquids up until two hours before anesthesia is no greater in acidity or volume than that of children who fast overnight. Therefore, ingesting clear liquids up to two hours before an operation does not appear to increase anesthetic risk. Because substantial physiologic benefit from a shortened fast has not yet been demonstrated, children with esophageal or gastric disease, severe neurologic disease, suffering from pain, receiving narcotics, or presenting airway management difficulties would be likely to benefit from more conservative fasting guidelines. In addition, until more data become available, infants younger than 1 year should fast for three to four hours before a surgical procedure.

The "shot to calm you" is understandably unpopular with children and parents alike. Oral, rectal, or nasal administration of the water-soluble benzodiazepine midazolam hydrochloride is an effective and painless alternative to intramuscular administration. Sedative doses of midazolam when given by these routes range between 0.5 and 1.0 mg per kg. About half of the drug is bioavailable following oral or rectal administration, and the maximal sedative effect will occur within 20 to 30 minutes. Administering the drug nasally increases bioavailability (to about 75%) and provides an even more rapid onset. Because the effects generally dissipate within an hour, recovery from anesthesia is not appreciably prolonged. Oral ketamine hydrochloride, 5 to 10 mg per kg, can be substituted for midazolam; some children may experience unpleasant psychomimetic effects, however, and recovery may be prolonged.

An oral transmucosal form of fentanyl citrate (Oralet) is now available. This consists of a medicated lozenge on a plastic holder. The dose of fentanyl (200 to 400 μg) in each Oralet is substantial, mandating strict adherence to administration and monitoring precautions.

In recent years the preoperative preparation of children for anesthesia and surgery has been reevaluated. Less stringent preoperative fasting guidelines and improvements in preoperative medication techniques have resulted in a less traumatic anesthetic experience without compromising safety.

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Inhaled Nitric Oxide

TREATMENT OF pulmonary hypertension with intravenous vasodilators is limited by systemic hypotension (due to the systemic vasodilator effects of all pulmonary vasodilators) and hypoxemia (due to the reversal of hypoxic pulmonary vasoconstriction). Inhaled nitric oxide (NO) now appears to be a major advance in the therapy for pulmonary hypertension.

Vascular endothelium is able to modulate vascular tone by producing substances that dilate the adjacent smooth muscle. In 1987 NO was identified as endothelium-derived relaxing factor. Nitric oxide produced by the endothelium diffuses into vascular smooth muscle, where it activates soluble guanylate cyclase; the subsequent increase in levels of intracellular cyclic guanine monophosphate produces smooth muscle vasodilation. Endothelium-independent nitrovasodilators such as nitroglycerin and sodium nitroprusside also activate guanylate cyclase, but do so by directly releasing NO. They release NO into both the pulmonary and systemic circulations, so that systemic vasodilation accompanies the pulmonary vasodilation. Nitric oxide itself is rapidly inactivated by hemoglobin in blood, so that the effect of inhaled NO may be localized to the lungs. Thus, inhaled NO diffuses from the alveoli to pulmonary vascular muscle and produces pulmonary vasodilation but no systemic effects. Although NO as a component of air pollution has been considered a toxic gas, it has relatively low toxicity; nitrogen oxides such as nitrogen dioxide that form from NO over time are polluting toxic compounds.

In animals, inhaled NO (5 to 80 parts per million) reverses pulmonary hypertension produced by global hypoxia, thromboxane-mimetic infusion, or heparin-protamine interactions. The pulmonary vasodilation is rapid, completely reversible, and selective, with no